CLINICAL CONSULTATION

The Clinical Consultation section is offered as an educational exercise under the direction of Etty Bitton, BSc, OD, Ecole d'Optométrie, Université de Montréal. It is intended to be a continuing forum wherein we as optometrists share our views and opinions on clinical topics with our colleagues. In this issue Etty Bitton, OD provided the case report. Yvon Rhéaume, OD and Richard Trevino, OD provide their views on how to manage the case reported below.

CASE REPORT

A Case of Unilateral Elevation in Intraocular Pressure

A 36-year-old African male consulted for an eye examination because of decreased vision. Ocular and medical histories were unremarkable. The patient is taking Privine (10 mg/day) for his allergies. Refraction revealed a slight myopia accompanied by oblique astigmatism (OD -0.50/ -0.50 x 035 VA 6/12; OS -0.50/-0.50 x 155 VA 6/ 7.5, OU $6/6^{-2}$). No improvement in visual acuities was found using a pinhole.

The anterior segment of the right eye was unremarkable. The anterior segment of the left eye post-dilation is depicted in Figure 1. Intraocular pressures measured by Goldmann tonometry were 11 mmHg OD and 19 mmHg OS. Fundus examination revealed slight asymetries in cup-to-disc (C/D) ratios. The right eye revealed a C/D ratio of 0.3/0.3 (H/V) while the left demonstrated a ratio of 0.3/0.4. The patient failed the Ishihara test and the D-15 test revealed a deutan-like anomaly. Central visual fields performed with the Humphrey Field Analyser revealed general reduction in sensitivity in both eyes.

QUESTIONS

1. Discuss the cause of the unilateral elevation in IOP in this patient.
2. What additional tests would you recommend, if any?
3. Discuss the management options for this patient.

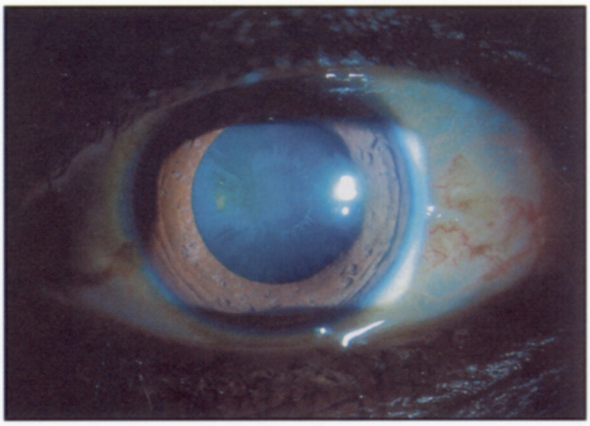

Fig. 1 Anterior segment of the left eye post dilation

Yvon Rhéaume, OD, Montreal, Quebec

Figure 1 clearly shows that this patient has pseudoexfoliation syndrome (PXFS) in the left eye. Exfoliation material on the anterior lens capsule is distributed in a classic pattern: a central zone or disc, an intermediate clear zone, and a peripheral granular zone. In the intermediate clear zone pupillary exfoliation material is brushed away by pupillary constriction and dilation. Consequently, deposits of exfoliation material become visible on the iris sphincter and pupillary margin and constitute a hallmark of PXFS in the predilation state. In PXFS there often is loss of pigment from the epithelium in the region of the iris sphincter; we now believe

that this pigment loss is caused by the rubbing of the exfoliation material against the iris while it is being dislodged, and the material then is dispersed throughout the anterior segment. Because of this, PXFS can be regarded as a pigment dispersion syndrome.

Exfoliation material is also deposited on the zonules and ciliary body, the corneal endothelium, the iris surface, and in the trabecular meshwork. It is now known that this material is provided by the equatorial lens epithelium, the iris pigment epithelium, and the nonpigmented ciliary epithelium. The deposition of this material causes alterations in the physiology of the tissue where it accumulates and obviously, at the trabecular meshwork level, along with the pigment dispersion, it obstructs the aqueous outflow system. In fact, PXFS is the single most common identifiable cause of open-angle glaucoma in the world.

In the present case, although the intraocular pressure (IOP) in the left eye is well within normal limits, the fact that there is such a difference in IOP between the two eyes should alert the clinician that there probably is some obstruction in the aqueous outflow system of the left eye, and thus the patient should have a water provocation test. I would expect that the test results will demonstrate a difference in IOP between the two eyes and thus a difference between the aqueous outflow system of each eye.

This young patient eventually will require IOP-lowering medication; the question is when. A referral to a glaucoma specialist is indicated.

Richard Trevino, OD, Kaiser Permanente Medical Center, Woodbridge, Virginia

Firstly, this case report can be summarized as follows: A 36-year-old African male presents with complaints of reduced vision. His history is unremarkable other than that he is taking a nasal decongestant for allergy. Best corrected visual acuity is 20/40 (6/12) and 20/25 (6/7.5) in the right and left eye, respectively. Intraocular pressure (IOP) is markedly asymmetric at 11 mmHg OD and 19 mmHg OS. Color vision is abnormal. Biomicroscopy reveals exfoliation material on the left lens capsule. Ophthalmoscopy reveals minimal asymmetry of the optic discs. Perimetry yields slight depression of the inferior nasal quadrants of each eye that fails to achieve statistical significance.

The clinical evaluation at this point would be directed at determining the cause of the reduced vision in the patient's eye and further evaluating the asymmetric IOPs.

Poor vision in a healthy appearing eye could be due to amblyopia, an afferent pathway lesion, or subtle retinal disease.[1] Oculomotor evaluation including cover testing should be performed to detect any strabismus that could be producing an amblyopia. This patient's visual fields have effectively ruled out a neurologic cause for the vision loss. Macular function should be evaluated with the Amsler grid and foveal threshold testing using an automated perimoter. Careful inspection of the macular region using the biomicroscope and Hruby or Volk lens would reveal any evidence of maculopathy. The general reduction in sensitivity of this patient's visual fields taken together with his color vision abnormality raises the remote possibility of a more widespread retinal dysfunction, such as might be caused by a retinal dystrophy.[2] The history could also be helpful in sorting out the cause of the patient's vision loss. If prior examinations have documented stable vision, the cause is likely functional. If, on the other hand, the vision loss is progressive, an aggressive search for its cause must be pursued.

When presented with asymmetric IOPs that are both within normal limits, the question becomes: Which eye has the abnormal pressure? The causes of unilateral lowering of IOP include anterior uveitis and retinal detachment.[3] The causes of unilateral elevation of IOP include primary angle-closure and all forms of secondary glaucoma, including exfoliation syndrome. The finding of exfoliation material in the eye with the higher IOP but not in the fellow eye does account for the asymmetry in pressures in this patient; however, it would still be prudent to inspect the anterior chamber angles with gonioscopy to confirm the absence of angle-closure glaucoma and other lesions.

The management of this patient's vision loss will depend upon its cause. If either no ocular abnormality is uncovered or it is deemed to be functional in nature, then no specific treatment is required. If the patient does indeed have a retinal lesion, such as central serous choroidopathy, then consultation with a retinologist for a fluorescein angiogram would be indicated if either the diagnosis is uncertain, a choroidal neovascular membrane is suspected, or laser treatment is to be instituted.[3]

This patient should also be managed as a glaucoma suspect, which entails periodic monitoring of the IOP, optic disc, and visual field. His risk factors for the development of glaucoma include the presence of exfoliation syndrome[4] and ethnic origin.[5] He should also be questioned about any family history of glaucoma.[6] I would first obtain several tonometry readings at vari-

ous times of day to determine if he has significant diurnal variation. If the tonometry readings remain at the same level as reported, then I would follow the patient with IOP monitoring every six months and annual perimetry. If his IOPs are found to rise significantly, I would monitor with tonometry and perimetry every six months. Inspection of the nerve head for splinter hemorrhages would be performed at every visit, and an annual dilation would be performed for reassessment of the cup-to-disc ratio and inspection of the nerve fiber layer. Indications for treatment would be a reproducible visual field defect, the presence of splinter hemorrhages on or near the optic nerve, or IOP exceeding 25 mmHg.

REFERENCES

1. Grala PE. Substandard acuity in the healthy looking eye. J Am Optom Assn 1981; 52: 725-729.
2. Cavender JC, Ai E, Lee ST. Hereditary macular dystrophies. In: Tasman W, Jaeger EA, eds. Duane's clinical ophthalmology. Philadelphia: Lippincott, 1993.
3. Cullom RD, Chang B, eds. The Wills eye manual. 2nd ed. Philadelphia: Lippincott, 1994.
4. Tarkkanen A. Exfoliation syndrome. In: Fraunfelder FT, Roy FH, eds. Current ocular therapy. 3rd ed. Philadelphia: Saunders, 1990: 543-545.
5. Tielsch JM, Sommer A, Katz J, et al. Racial variations in the prevelance of primary open-angle glaucoma. JAMA 1991; 266: 369-374.
6. Tielsch JM, Katz J, Sommer A, et al. Family history and risk of primary open-angle glaucoma. Arch Ophthalmol 1994; 112: 69-73.

Radiology Basics for Primary Eye Care

(continued from page 180)

and contraindications are essential for the detection and evaluation of certain ocular anomalies in primary eye care.❑

REFERENCES

1. Squire LF. Fundamentals of roentgenology. Cambridge, MA: Harvard University Press, 1971: 1.
2. Tower H, Oshinskie LJ. An introduction to computer tomography and magnetic resonance imaging of the head and visual pathways. J Am Opt Assoc 1989: 60(8): 619-628.
3. Oshinskie LJ. Radiology. In: Eskridge JB, Amos JF, Barlett JD , eds. Clinical procedures in optometry. Philadelphia: JB Lippincott, 1991: 580-595.
4. Grainger RG. Techniques and imaging modalities. In: Grainger RG, Allison DJ, eds. Diagnostic radiology, Vol. 1. New York: Churchill, 1986: 3-110.
5. Kolodny NH, Evangelos SG, D'Amico DJ, Albert DM. Magnetic resonance imaging and spectoscopy of intraocular tumor. Surv Ophthalmol 1989; 33(6): 502-514.
6. Hupp SL, Kline LB. Magnetic resonance imaging of the optic chiasm. Surv Ophthalmol 1991; 36(3): 207-216.
7. Dutton JJ. Gliomas of the anterior visual pathway. Surv Ophthalmol 1994; 38(5): 427-450.
8. Carter KD, Nerad JA. Plain roentgenographic evaluation of orbital disease. Vol. 2, Chapt. 23. In: Tasman WD, Jaeger EA, eds. Duane's clinical ophthalmology. Philadelphia: JB Lippincott, 1992: 1-14.
9. Saint-Louis LA, Haik BG, Amster JL. Magnetic resonance imaging of the orbit and visual pathways. In: Haik BG, ed. Advanced imaging techniques in ophthalmology. Int Ophthalmol Clin 1986: 26(3): 169-185.
10. Garrity JA, Forbes GS. Computed tomography of the orbit. Vol. 2, Chapt. 24. In: Tasman W, Jaeger EA, eds. Duane's clinical ophthalmology. Philadelphia: JB Lippincott, 1992: 1-16.
11. Talley DK. Clinical laboratory testing for the diagnosis of systemic disease associated with anterior uveitis. Optom Clin 1992; 111.
12. Dutton J, Slamovitis. Management of blowout fracture of the orbital floor. Surv Ophthalmol 1991; 35(4): 279-291.
13. Tang RA, Kramer LA, Shiffmann J, et al. Chiasmal trauma: Clinical imaging and considerations. Surv Ophthalmol 1994; 38 (4): 381-383.
14. Wormington CM. Pituitary adenoma: Diagnosis and management. J Am Opt Assoc 1989; 60(12): 929-935.